COMPARISON OF VARIANCE AND COVARIANCE PATTERNS IN PARALLEL AND SERIAL THEORIES OF TIMING

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Parallel and serial timing processes are analyzed for their account of the dynamics of intertrial responding in the peak procedure. A strictly serial model, such as the behavioral theory of timing (Killeen & Fetterman, 1988), does not fit the dynamic correlation pattern in the location and duration of the middle high-rate responding portion of peak trials. In contrast, the parallel scalar expectancy theory model, with a sample for memory and threshold, does fit this pattern. A modification of the serial model is presented that also accommodates the within-trial covariance pattern. The modification, which is formally equivalent to a model for human tapping (Wing & Kristofferson, 1973), entails the addition of concurrent processes operating in parallel with serial timing.

Key words: scalar expectancy, behavioral timing, peak procedure, parallel and serial processing

Much past theoretical effort has been devoted to understanding a variety of phenomena in animal learning in terms of molar conceptions of the processes underlying the behavior. Central to this effort has been the characterization of responding as adapted to the time and conditions of reinforcement. Two largely successful yet conceptually different treatments of the static features of adjustment to the time of reinforcement have been scalar expectancy theory (SET; Gibbon, 1977; Gibbon & Church, 1990) and an alternative approach by Killeen and Fetterman (1988), the behavioral theory of timing (BeT). The purpose of the present report is to cast these theories in their most general form and contrast their treatment of the within-trial dynamics of a timing performance.

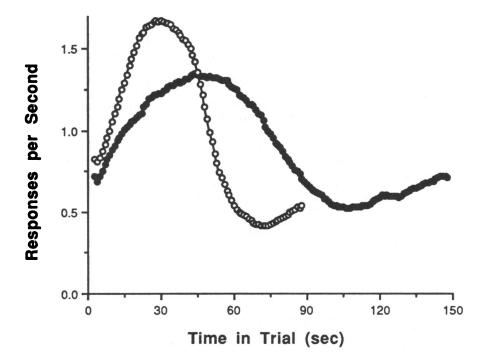
According to SET, timing processes involve monitoring current time and contrasting it with memory for times that have been associated with reinforcement in the past. The monitoring of current time (clock process), the memory process by which subjects "know" the target time, and the decision process ensuring a conservative behavioral strategy with respect to the contingencies of reinforcement are concurrent processes. They are cognitive processes in the sense that continuous processing is assumed that is not reflected in behavior until criterion decision thresholds are met. In contrast, Killeen and Fetterman (1988) argue that

such processes are not really necessary. BeT is characterized as parsimonious in not requiring the memory, detection, and threshold processes embodied in SET. It posits a simple system in which a Poisson process generates successive states, some of which become associated with reinforcement. Although the mechanism (and degree) of association are often described with a rather broad brush, the central thrust of this system is sufficient to account for much timing data.

SET and BeT differ in at least two important ways. First, they differ with respect to their explanation of what we have called the scalar property—a Weber law-like feature of timing data that arises in a wide variety of settings. It may be described empirically as a relativistic property of time-based performance that results in a common form for the performance measure when different absolute times are normalized. That is, the data superpose when plotted in relative time.

According to scalar expectancy theory (cf. Gibbon, in press; Gibbon & Church, 1984, 1990), this property may arise from several different sources of variance in the processing system that records, remembers, and compares times. The common feature of these scalar sources is that they act multiplicatively on the representation of reinforcement time. In contrast, the behavioral theory of timing accounts for the scalar property by a change in the rate of the Poisson pacemaker underlying successive state changes. The pacemaker is controlled by the overall rate of reinforcement in the situation, and runs at a speed proportional to this rate. We believe that this difference between

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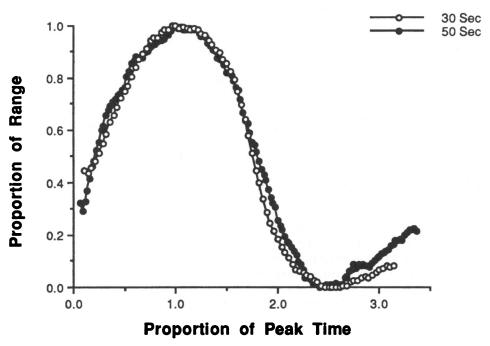


Fig. 1. Peak functions averaged over 4 birds studied at 30-s and 50-s peak procedures. The top panel shows data in absolute time, T; the bottom shows normalized data plotted relative to time of the peak, T^* . Normalization occurred in two steps. First each subject's peak function was smoothed to obtain maximum and minimum rates and time of the maximum (peak time). Then each function was taken as a proportion of its range, $R/(R_{\text{max}} - R_{\text{min}})$ and plotted at time relative to peak time (T/T^*) , before averaging across subjects. This transformation controls for individual differences in peak times and maximum and minimum (operant level) rates. Data are from the experiment reported in part by Gibbon and Church (1990).

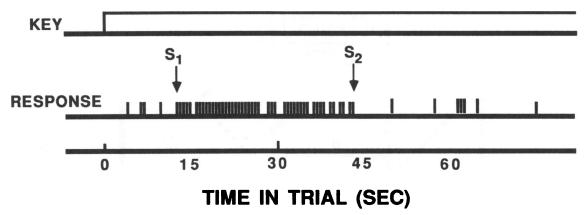


Fig. 2. A sample trial from the 30-s peak procedure. Responding tends to be bunched around the time of reinforcement. The beginning (s_1) and end (s_2) of the high-rate period are indicated with arrows.

the two accounts is testable, but it is not our purpose to do so here. Rather, we will concentrate on a second dynamic dimension along which the accounts differ.

Parallel versus Serial Processing

A second difference between the theories is that SET is a parallel model, whereas BeT is a serial model. We contrast the parallel and serial approaches with their treatment of the peak procedure. In the peak procedure, responding is reinforced on some percentage of trials (food trials) after a given fixed interval has elapsed from the signaled onset of the trial. On other trials ("peak" or "nonfood" trials), the signal simply stays on for a long period of time and no food is presented. The procedure was originally devised by Catania (1970) and was subsequently studied by Roberts (1981), Meck and Church (1984), and others (e.g., Gibbon & Church, 1990). Responding on peak trials tends to occur around the time when reinforcement would be due on food trials, and is roughly symmetric on either side of this peak time (though often slightly skewed to the right; cf. Gibbon, Church, & Meck, 1984). An example taken from Gibbon and Church (1990) shows peak functions pooled over 4 birds studied under two conditions, a 30-s and 50-s peak procedure in which responding was reinforced at the fixed interval on half the trials. The other half of the trials lasted for three times the fixed interval, and no food was given. Responding increased to a peak at about the time of reinforcement and declined in a roughly symmetric manner after that time, as seen in the top panel of Figure 1. The two functions approximately superpose when normalized and plotted in time relative to the time of the peak rate, as shown in the bottom panel. This is the scalar property.

Although responding appears to follow a smooth transition through the peak, Gibbon and Church (1990) analyzed the behavior in individual trials as a two-state process. Responding occurred at a low rate in State 1, changed to a high rate at some point prior to the time of reinforcement, and changed back again to a low rate at some time after the reinforcement time. An example from a 30-s peak trial is shown in Figure 2. At time zero, the keylight is turned on. Responses are infrequent until about Second 15, and then occur rather bunched around the time of reinforcement, dropping to a low rate after about Second 45. Indicated in the figure is a start (s_1) and stop time (s_2) for the period of high-rate responding. We argued that the smooth bellshaped peak functions from this procedure are produced by a discrete two-state process, but one that has variable locations and durations on different trials. Averaging these individual trial functions produces the smooth curve. The idea is an extension of Schneider's early, seminal "break-run" analysis of fixed-interval performance (Schneider, 1969). We simply add a subsequent break back to a low rate on peak trials when reinforcement is not forthcoming.

Gibbon and Church (1990) presented an analysis of break-run-break patterns in individual trials. The start and stop times on each trial were obtained from a least squares fit of

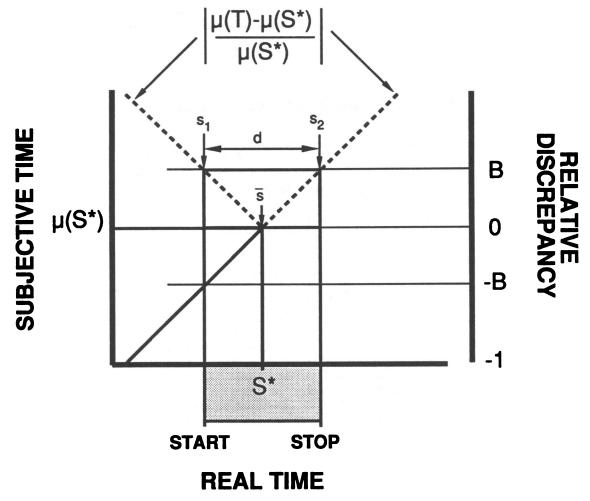


Fig. 3. Schematic representation of the linear time sense assumed in SET. Subjective time accumulates linearly in real time, and the high-rate state begins when the accumulation crosses a lower threshold (-B on the relative scale) and continues until it crosses an upper threshold (+B). The right-hand ordinate represents the relative discrepancy between the representation of the current time and the reinforced time. The dashed function is the absolute value of the relative discrepancy (right-hand ordinate). In this example, all values are taken at their average [μ (.)]. On the bottom axis, the stippled area represents the high state of responding (adapted from Gibbon & Church, 1990, Figure 10).

three horizontal line segments to response-rate data in individual time bins. The reader is referred to Gibbon and Church (1990) for further elucidation of the method. It is useful for this analysis to define for each trial four statistics obtainable from the break-run-break pattern: the time of the start (s_1) , the time of the stop (s_2) , the spread (d, the duration of the high state between s_1 and s_2), and the middle $(\bar{s},$ the arithmetic center of the high state). These measures are shown in the schematic representation in Figure 3.

Application of SET to the peak procedure. According to scalar expectancy theory, subjective time increases linearly during a trial, as in the diagonal function of Figure 3. Subjects monitor time during the trial and begin responding (that is, go into the high-response state) when the subjective representation of the current time is close enough to their memory for the reinforced time. The memory for time of reinforcement here is taken as $\mu(S^*)$, and responding begins when the current time becomes greater than a threshold value (-B on

the relative scale). Subjects continue high-rate responding until that threshold is exceeded on the far side of the remembered reinforcement time (+B). An alternative representation is shown with the dashed function referred to the relative discrepancy axis on the right. This function plots the absolute value of the discrepancy between the current time, $\mu(T)$, and the remembered time, $\mu(S^*)$, relative to $\mu(S^*)$. On the lower axis, the start and stop time bracketing reinforcement time is shown. The theory argues that variability in the speed with which the current time is accumulated, in the memory for the reinforcement time and in the threshold for responding, may all contribute to produce a variety of start and stop times on different trials. (A continuous version of start and stop times on each trial might identify inflection points in a smooth rise to a high rate and a subsequent fall to a low rate. For our present purposes either characterization will do, although we believe, and the data confirm, that the two-state process is a better first-order description of individual-trial performance.)

To summarize, at the outset of each trial, subjects sample from their memory for the reinforcement time, and during the elapsing interval, contrast this time with their representation of the current time. They respond at a high rate when their memory of the reinforced time and their representation of current time are "close enough" to each other by a threshold criterion. Hence at least three parallel processes—memory search, monitoring of current time, and selection of a response threshold—are concurrently undertaken in such a way that a comparison between past and present, so to speak, is continuously available.

Application of BeT to the peak procedure. The behavioral theory of timing, and indeed any strictly serial renewal process, is to be contrasted with the parallel system in that decisions are made singularly and successively. The general serial-process approach to this performance would first determine a start time, then a duration for the high rate of responding, followed in turn by the low state. As an example, consider the following adaptation of the Killeen-Fetterman (1988) account for this situation. We imagine that over a large number of training trials, reinforcement has impinged upon a subset of m states (we will call them states n + 1 through n + m) turning them into "hot" states, engendering high response

Table 1
Variance and covariance values for within-trial statistics from Gibbon and Church (1990).

	<i>s</i> ₁	<i>s</i> ₂	d	š
Start, s ₁	86.10	·····		
Stop, s ₂	32.30	114.86		
Spread, d	-48.05	77.12	127.13	
Middle, š	56.85	72.20	15.04	68.70

rates. This adaptation of their account is of course ad hoc, and we simplify it further by assuming that between the first reinforcementassociated state and the last reinforcement-associated state, there exist no states not associated with reinforcement. Over long training this will be true but, of course, over long training it will also true be that all states in the process are associated with some reinforcement. Hence our adaptation really requires an implicit threshold for high-rate responding. If a state has been associated with reinforcement sufficiently often (or, perhaps, with a sufficiently high probability) it becomes a highrate state, and there is a collection of these spanning the middle range around reinforcement, as shown in Figure 4.

We assume that there exists such an unspecified threshold, identifying state n as the transition point from low to high rates, and identifying state n + m as the last hot state. The first n states do not occasion high-rate responding, the next m states do, and thenceforth (from n + m + 1 on) responding again resumes a low rate. Under these assumptions, the distribution of start times is gamma (nfold), the distribution of stop times is gamma (n + m-fold), and the distribution of spreads, the duration of the high state, is also gamma (m-fold). Given n and m greater than about five, these forms are compatible with the data distribution forms. They are roughly symmetric, but have some skew, with the distribution of s_1 more skewed than that of s_2 .

Intertrial Dynamics

Average variance and covariance patterns from the peak experiment reported in part in Gibbon and Church (1990) are presented in Table 1 for the four measures. Several features of these data are diagnostic for a discrimination between parallel and serial models. First, the variances (main diagonal) show that the vari-

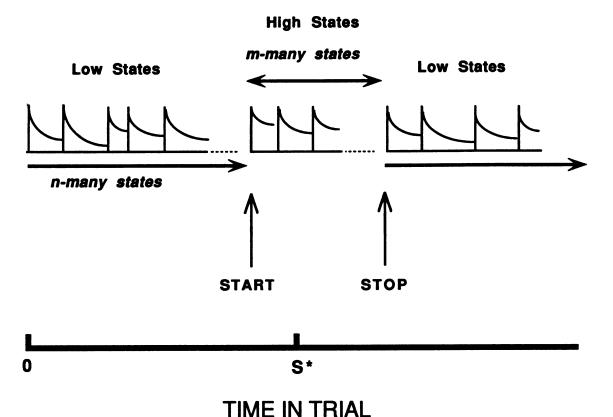


Fig. 4. Schematic representation of BeT for the peak procedure. The Poisson process initiates successive states with random, exponentially distributed durations. It is assumed that after n-many of these, a set of m-many "high" states ensues during which subjects respond at a high rate, followed in turn by a low rate in succeeding states.

ance of s_2 is greater than s_1 and the variance of d is greater than \bar{s} . The covariance pattern shows a positive start-stop covariance and a negative start-spread covariance. All subjects showed this pattern. The other covariances are less diagnostic but are included for completeness. We will concentrate here on the startstop and start-spread values.

Variance. The variance patterns expected for the parallel model (SET) and the general serial model are shown in Table 2. A memory sample and a threshold sample are assumed for SET, and determinations of t (start time or s_1) and of h (hot time or high-rate duration, d, ending in s_2) are assumed for the serial model. These correspond to the first n states and the next m states in BeT.

The memory sample, x, for the parallel model is assumed to have a mean close to the reinforcement time, $E(x) = \mu(S^*)$, and variance, σ_x^2 . The threshold sample b, which is

independent of the memory, has a mean B and variance σ_b^2 . From Figure 3 we can

see that the lower threshold is reached when
$$\left| \frac{\mu(s_1) - x}{x} \right| = b$$
, or $(1 - b)x = \mu(s_1)$.

Similarly, the upper threshold for stopping responding is met when $(1 + b)x = \mu(s_2)$. This produces a variance pattern in which s_1 , s_2 , and d reflect both threshold and memory variance, but \bar{s} does not. The midpoint between s_1 and s_2 reflects only memory variance. When the lower and upper thresholds depend on the same sample, they cancel in the middle statistic. (The variance pattern is unchanged if one permits an additional constant, say Δ , to enter into one of the thresholds allowing an asymmetric peak time $\neq S^*$.)

The variance pattern for the serial model is shown on the right in Table 2. In contrast to the parallel model, the serial model has a very

Table 2. Variance Patterns for Parallel and Serial Models

	Parallel (SET)	Serial
Start	$\sigma_{b}^{2} (\sigma_{x}^{2} + S^{2}) + (1-B)^{2} \sigma_{x}^{2}$	σ _t ²
Stop	$\sigma_{b}^{2} (\sigma_{x}^{2} + S^{2}) + (1+B)^{2} \sigma_{x}^{2}$	$\sigma_{t}^{2} + \sigma_{h}^{2}$
Spread	$4\sigma_{b}^{2}(\sigma_{x}^{2}+S^{2})+4B^{2}\sigma_{x}^{2}$	σ _h ²
Middle	σ _x ²	$\sigma_{t}^2 + \frac{1}{4}\sigma_{h}^2$
Constraints:	$\sigma_{s_2}^2 > \sigma_{s_1}^2$	$\sigma_{s_2}^2 > \sigma_{\bar{s}}^2 > \sigma_{s_1}^2$ $\sigma_{s_2}^2 > \sigma_{d}^2$ $\sigma_{s_2}^2 = \sigma_{s_1}^2 + \sigma_{d}^2$ Note: For BeT, $\sigma_{t_1}^2 = n\mu^2$ and $\sigma_{h_1}^2 = m\mu^2$, where $\mu = \text{mean state residence time}$

simple form, with the sample for the start time being the only contributor to variance of s_1 , and the sample for the high-state duration being the only contributor to variance of the spread. The other two statistics, stop time and middle, reflect the sum of both variables.

Below the variance listings are some qualitative and quantitative predictions entailed by the parallel and serial models. For the parallel model, all that may be concluded with certainty from the variance pattern is that stop variance must be greater than start variance. For the serial model, several additional constraints are available. Stop variance must exceed start variance, and the variance of the middle must lie between these two. Moreover, stop variance must exceed the variance of the spread. A considerably stronger prediction is available for this model as well: The variance of the spread must be precisely the difference between stop and start variance, because start and spread are determined successively and independently.

Figure 5 shows variance patterns for indi-

vidual birds from the experiment reported in part in Gibbon and Church (1990). Note that stop variance does exceed start variance as required by both models, but the variance of the middle does not lie between these two. Rather, it is less than either. The stronger constraint, implied by the independence of the initial low state and the high state for the serial model, also is not met by these data. The prediction for the stop variance is shown by the dark bar (\hat{s}_2). Stop variance should be simply the sum of the start and spread variance, and it is clear that the obtained s_2 variances fall below this value. This is due to the negative covariance between start and spread, analyzed next.

Covariance. The pattern of covariation among the four measures is a still more powerful discriminator between these models. We will briefly recapitulate the central features of the correlation patterns between start and stop and start and spread (the major diagnostic syndrome) for the parallel SET model. The reader is referred to Gibbon and Church (1990) for a more extensive treatment. In Figure 6 the

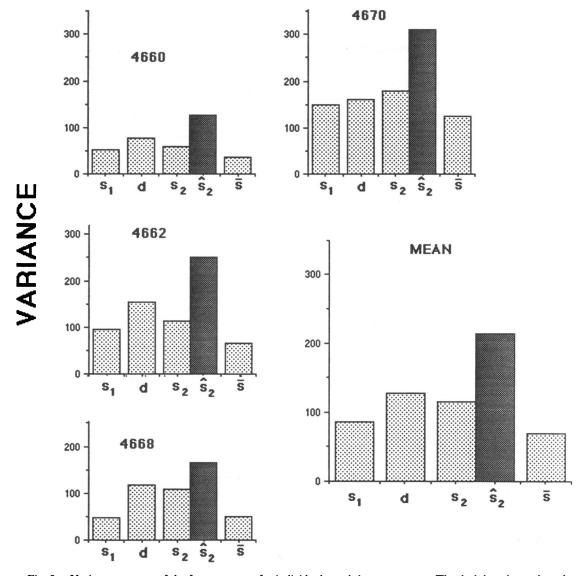


Fig. 5. Variance patterns of the four measures for individuals, and the group mean. The dark bar shows the value of stop variance predicted by the serial model (\hat{s}_2) .

pattern expected from variation in memory alone is shown on the left, for successive trials in which samples from memory vary from shorter than the reinforcement time to longer than the reinforcement time. The threshold is assumed to be constant. The mechanism described in Figure 3 produces, as may be seen, a series of broader high states as the memory sample increases. Increases in the estimate of reinforcement time induce both a later start and a later stop. It is readily seen that this entails a perfect positive correlation when

memory variance is the only contributor to variability.

On the right side of Figure 6, we show the pattern expected when the memory contributes no variance and threshold contributes all the variance. Here the middle remains constant (set here at reinforcement time), but start and stop are perfectly negatively correlated. Varition in the threshold that produces a conservative (early) start time produces a corresponding conservative (late) stop time and vice versa. Variance in memory and variance in threshold

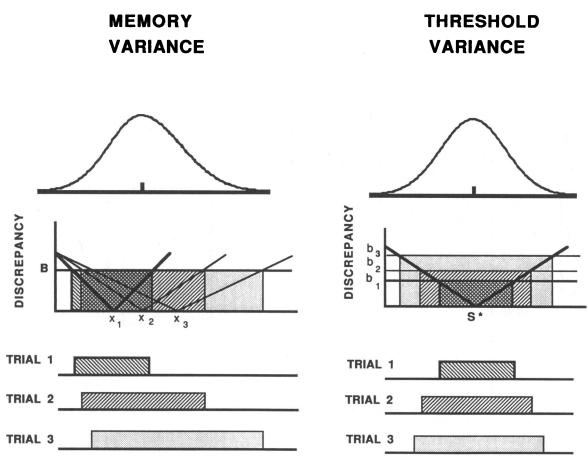


Fig. 6. Schematic representation of start, stop, and spread durations on successive trials for SET. On the left, memory estimates of reinforcement time vary (x_1, x_2, x_3) . Increasing estimates induce increasing start and stop times and increasing spreads. On the right, memory estimates are assumed constant at reinforcement time, and variation is induced by different thresholds (b_1, b_2, b_3) . Threshold variation induces negative covariation between start and stop times, as well as between start and spread.

work in opposite directions for the correlation pattern, with memory inducing a positive s_1, s_2 correlation and threshold inducing a negative s_1, s_2 correlation.

The correlation pattern for s_1 and spread, d, is essentially the same. Memory variance induces a positive correlation between s_1 and d—late starts are accompanied by long hot times. Threshold variance induces a negative correlation between start and spread—late starts produce shorter hot times. The two sources of variance again work in opposite directions for the expected correlation pattern. More important, however, it may be shown that the s_1, s_2 positive correlation is more robust than the s_1, d positive correlation for memory variance. Correspondingly, the s_1, s_2 negative

correlation induced by threshold variance is less robust than the s_1 ,d negative correlation induced by this source of variance. Hence, when both sources of variance are operating together, the correlation pattern expected is moderate positive for s_1 , s_2 and moderate negative for s_1 ,d. The variance and correlation forms are derived in the appendix, where this pattern is analyzed quantitatively.

This pattern is not expected from the serial model. In the serial model, other things being equal, a late start produces a late stop. Because the sample for the hot time is determined independently from the random variable determining starts, a late start simply adds time to the average stop time, and hence a positive s_1, s_2 correlation is expected. More important, how-

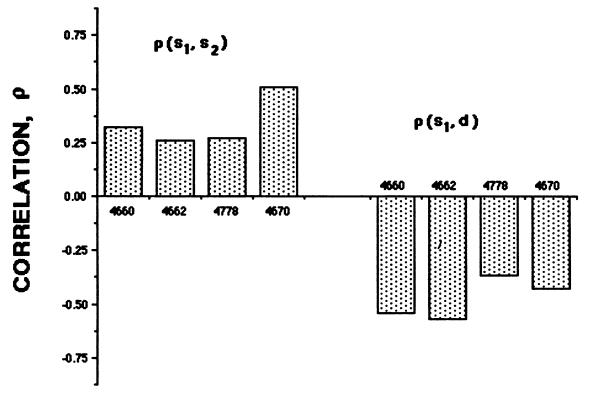


Fig. 7. Correlation patterns for individual birds. Start, stop correlations, $\rho(s_1, s_2)$, are on the left, and start, spread correlations, $\rho(s_1, d)$, are on the right.

ever, the start-spread correlation expected from a serial model is in fact zero, because the two processes are independent.

In Figure 7 the start-stop and start-spread correlations for the same birds are shown. In every case the start-stop correlation is positive and the start-spread correlation is negative.

To summarize thus far, the variance/covariance data do not support the general serial model with two random samples taken in succession for start and duration of the high state. The data pattern is accommodated, however, by the parallel model, SET, with two samples, one from the memory for the target, time of reinforcement, and a second for a threshold used to determine when to enter and exit the high-response state.

An Extension of the Serial Model

In the conference that this issue memorializes, Gibbon presented a way to salvage the general serial model, though at some cost. The method transforms the serial model into a half parallel, half serial, or if you will, quasi-serial

model by adding concurrent processing stages after the determination of the trigger to start the high state and the trigger to stop the high state. These induce lags in the actual start and stop times, comprising a third and fourth random variable.

This is a classical time-series model (Box & Jenkins, 1970; Glass, Willson, & Gottman, 1975). It was developed by Wing and Kristofferson (1973) for serial tapping performances in humans, and was independently rediscovered by Pittendrigh and Daan (1976) in a circadian timing model in which the lag between the start time and responding was taken as a "wake-up" time for activity-rest cycles. It has also been used to account for the interlickinterval distribution of rats drinking and the interresponse-interval distribution of humans tapping in synchrony with a metronome (Church, Broadbent, & Gibbon, in press). The model is shown in schematic form over the right-hand column of Table 3. Here time t initiates a random delay—we may call it an instructional delay (i_1) —before the behavioral

	Serial	Quasi-serial
	t h 0 s ₁ s ₂	
Start	σ_i^2	$\sigma_{i^{2}+}^{2}$ $\sigma_{i_{1}}^{2}$
Stop	$\sigma_{t}^{2} + \sigma_{h}^{2}$	$\sigma_t^2 + \sigma_h^2 + \sigma_{i_2}^2$
Spread	σ _h ²	$\sigma_{h}^{2} + \sigma_{i_{1}}^{2} + \sigma_{i_{2}}^{2}$
Middle	$\sigma_{t}^{2} + \frac{1}{4} \sigma_{h}^{2}$	$\sigma_{i}^{2} + \frac{1}{4} (\sigma_{h}^{2} + \sigma_{i_{1}}^{2} + \sigma_{i_{2}}^{2})$
ρ(s ₁ ,s ₂)	$\left(1 + \frac{\sigma_h^2}{\sigma_t^2}\right)^{-1/2}$	$\left[\left(1+\frac{\sigma_{i_{1}}^{2}}{\sigma_{i_{1}}^{2}}\right)\left(1+\frac{\sigma_{h}^{2}}{\sigma_{i_{1}}^{2}}+\frac{\sigma_{i_{2}}^{2}}{\sigma_{i_{1}}^{2}}\right)\right]^{-1/2}$
ρ(s ₁ ,d)	0	$-\left[\left(1+\frac{\sigma_{t}^{2}}{\sigma_{i_{1}}^{2}}\right)\left(1+\frac{\sigma_{t}^{2}}{\sigma_{i_{1}}^{2}}+\frac{\sigma_{i_{2}}^{2}}{\sigma_{i_{1}}^{2}}\right)\right]^{-1/2}$

Table 3. Variance and Correlation Pattern for Serial and Quasi-Serial Models

consequence of high-rate responding is initiated. Concurrently, in parallel with this, the spread determination is unfolding, and at its termination at time t+h, a second instructional lag (i_2) ensues before the cessation of responding. The second lag we might call the "go-to-sleep time" before behavior indeed sleeps in the final low state.

Notice that the original serial model has been removed, if you will, one layer below the behavior (perhaps encouraging some cognitive nomenclature). The instructional system that actually effects the behavior takes some time, and it is this time that induces a negative correlation between start and spread, via regression to the mean.

In Table 3 we present the variance and correlation pattern expected for the start-stop and start-spread correlations for both the serial model and the more complex quasi-serial model on the right. To do the job intended, the quasi-serial model must have i_1 and i_2 independent. A moment's reflection will show that if they are the same, albeit randomly varying from

trial to trial, then the correlation prediction for start-spread remains at zero. However, as long as independence holds between these two delays ensuing in parallel with the hidden layer, the correlations for start-stop and start-spread are qualitatively consonant with the data patterns. (The appendix describes quantitative limit behavior of these correlations that may be strained by some data patterns.)

This adjustment adds parallel processing stages to the original serial model with the concomitant intellectual cost in parsimony: The two-variable serial model becomes a four-variable quasi-serial model with means and variances for each variate. On the other hand, parsimony may not be the ombudsman of choice in a serious attempt to address a truly broad variety of dynamic as well as static response forms. For example, although on its face the quasi-serial model requires four independent random variables and hence eight parameters (plus a threshold, perhaps, for defining the high states), one might readily imagine that the instructions to start and stop induce delays

drawn from the same distribution, reducing the parameter set by two.

Also, although we have advertised SET as qualitatively accommodating these data, a good quantitative fit may require more than one threshold. Moreover, the degree to which the mean values enter into these autocorrelation functions has not been analyzed in its full extent. For example, Table 3 involves only variances for the quasi-serial model, whereas for the parallel model mean values (Table 2 and the appendix) enter as well because of their role in product variance calculations. Thus, dynamic details force at least some parallel processing, but the details of that kind of processing are not themselves forced. We cannot rule out a quasi-serial model.

An alternative model that is conceptually similar to the parallel model but formally similar to the quasi-serial model is instructive. Imagine a theory in which a sample from memory is chosen for the target time, but then a spread value is chosen independently from a different distribution, and with it some added random error at the beginning and the end. Such a model is conceptually close to SET but formally close to the quasi-serial model. Like the quasi-serial model, it induces a negative correlation between s_1 and d via regression to the mean. Again also, there is some cost in complexity because four variables seem to be required, although some reduction in this complexity may be afforded if the added errors are drawn from a single distribution.

Conclusion

SET is by nature a parallel processing structure. General renewal processes, and in particular BeT, in contrast, are by nature serial processing structures. They therefore do not predict the dynamic correlation patterns seen in the peak procedure. However, a modification of the serial model with partial parallel components is feasible and does accommodate the data.

In summary then, the variance and covariance patterns in the trial-by-trial dynamics of timing in the peak procedure require some kind of parallel processing mechanism underlying the central features of the data. We argue that this requirement takes these mechanisms back into hidden layers of processing often thought of as cognitive. Moreover, serial mod-

els in general must come to grips with the details of the mnemonic process whereby the high-rate states bracketing the target time are identified and remembered. Perhaps once such identifications are made, it will become moot whether we call these mechanisms cognitive or behavioral—they are certainly parallel.

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APPENDIX

Variance and Covariance

SET. According to SET the variances and covariances for the statistics we have identified are obtained from two independent samples of random variables. One sample, x, represents the estimate of the remembered target time. It has a mean, which we will assume to be centered on the true time, S*, and a variance, σ_x^2 . The second variable, b, is an independent sample from a distribution of threshold values centered on a mean, B (0 < B < 1), which determines when the current time is close enough to the target time to warrant high-rate responding. From Figure 3 we see that the lower threshold is met when $s_1 = (1 - b)x$ (assuming a negligible intercept in the linear subjective time function). Similarly, the upper threshold is met when $s_2 = (1 + b)x$. To obtain the variance of start and stop times, we use the well known relation for the variance of the product of two independent random variables,

$$Var(xy) = \sigma_x^2 \sigma_y^2 + \mu_x^2 \sigma_y^2 + \mu_y^2 \sigma_x^2, \quad (1)$$

(cf. Gibbon, 1977). Using the multiplication Rule 1 we have

$$Var(s_1) = \sigma_b^2(\sigma_x^2 + S^{*2}) + (1 - B)^2\sigma_x^2$$
 (2)

$$Var(s_2) = \sigma_b^2(\sigma_x^2 + S^{*2}) + (1 + B)^2\sigma_x^2, \quad (3)$$

as shown in the top two rows of Table 2. The difference, d, is $s_2 - s_1 = 2bx$ so its variance is

$$Var(d) = 4\sigma_b^2(\sigma_x^2 + S^{*2}) + 4B^2\sigma_x^2,$$
 (4)

by application of the same rule. The variance of the middle is simpler, as

$$\bar{s} = (\frac{1}{2})(s_1 + s_2) = x,$$

so that

$$Var(\bar{s}) = \sigma_{r}^{2}.$$
 (5)

The random variable, b, cancels in the sum of s_1 and s_2 so that the variance of the middle reflects only variance of the memory for the target time.

Equations 2 through 5 are the entries in the column for the parallel model, SET, in Table 2. The entries for the serial model follow immediately from the independence of the random variables determining the start $(s_1 = t)$, and the spread (d = h).

Correlations

 $\rho(s_1,s_2)$. For the determination of correlation, we use the well-known relationship between the variance of the sum of two random variables and their covariance:

$$Var(x + y) = \sigma_x^2 + \sigma_y^2 + 2Cov(x, y),$$

or

$$2\text{Cov}(x, y) = \text{Var}(x + y) - (\sigma_x^2 + \sigma_y^2).$$
 (6)

The covariance in turn is

$$Cov(x, y) = \rho(x, y)\sigma_x\sigma_y, \tag{7}$$

so that

$$\rho(x, y) = \frac{\operatorname{Var}(x + y) - (\sigma_x^2 + \sigma_y^2)}{2\sigma_x\sigma_y}.$$
 (8)

SET. It is convenient for the correlation calculations to express the three free parameters, σ_x^2 , σ_b^2 , and B, in terms of coefficients of variation. Let the lower (l) and upper (u) thresh-holds be defined as l = 1 - b and u = 1 + b, with means L = 1 - B and U = 1 + B and variance, σ_b^2 . Then define

$$\gamma_x \equiv \frac{\sigma_x}{S^*}, \quad \gamma_l \equiv \frac{\sigma_b}{L}, \quad \text{and} \quad \gamma_u \equiv \frac{\sigma_b}{U}.$$
(9)

The variance of the sum $s_1 + s_2 = 2\bar{s}$ is found from Equation 5 as simply $4\sigma_x^2$, or, using the conventions for U and L,

$$Var(s_1 + s_2) = \sigma_x^2(U + L)^2$$
.

Using U and L in the variances of s_1 and s_2 (Equations 2 and 3), the covariance formula, Equation 6, yields

$$2\text{Cov}(s_1,s_2) = \sigma_x^2(\mathbf{U} + \mathbf{L})^2 - 2\sigma_b^2(\sigma_x^2 + \mathbf{S}^{*2}) - \sigma_x^2(\mathbf{U}^2 + \mathbf{L}^2).$$

Collecting terms,

$$2\text{Cov}(s_1, s_2) = \sigma_x^2(2\text{UL}) - 2\sigma_b^2(\sigma_x^2 + S^{*2}).$$
(10)

Finally, using the right-hand side of Equation 10 in Equation 8, and dividing numerator and denominator by 2UL, we obtain the correlation in terms of the coefficients of variation, Equation 6:

$$\rho(s_{1},s_{2}) = \frac{1 - \gamma_{l}\gamma_{u}\left(1 + \frac{1}{\gamma_{x}^{2}}\right)}{\sqrt{\left[1 + \gamma_{l}^{2}\left(1 + \frac{1}{\gamma_{x}^{2}}\right)\right]\left[1 + \gamma_{u}^{2}\left(1 + \frac{1}{\gamma_{x}^{2}}\right)\right]}}$$
(11)

 $\rho(s_1,d)$. A similar development is used for the start-spread correlation implied by SET. Note that $s_1 + d = s_2$ so that

$$\begin{aligned} 2\text{Cov}(s_1,d) &= \sigma_b^2(\sigma_x^2 + \text{S*}^2) + \text{U}^2\sigma_x^2 \\ &- \sigma_b^2(\sigma_x^2 + \text{S*}^2) - \text{L}^2\sigma_x^2 \\ &- 4\sigma_b^2(\sigma_x^2 + \text{S*}^2) \\ &- (\text{U} - \text{L})^2\sigma_x^2, \end{aligned}$$

or

$$2\text{Cov}(s_1,d) = 2\sigma_x^2 L(U - L) - 4\sigma_b^2 (\sigma_x^2 + S^{*2}).$$
 (12)

For this correlation, we define a difference coefficient of variation, γ_{-} ,

$$\gamma_{-} \equiv \frac{2\sigma_{b}}{\mathbf{U} - \mathbf{L}} = \frac{2}{\frac{1}{\gamma_{c}} - \frac{1}{\gamma_{c}}}.$$
 (13)

Then using the right-hand side of Equation 12 in Equation 8, and dividing numerator and denominator by 2L(U - L), we have

$$\rho(s_1,d)$$

$$= \frac{1 - \gamma_{l} \gamma_{-} \left(1 + \frac{1}{\gamma_{x}^{2}}\right)}{\sqrt{\left[1 + \gamma_{l}^{2} \left(1 + \frac{1}{\gamma_{x}^{2}}\right)\right] \left[1 + \gamma_{-}^{2} \left(1 + \frac{1}{\gamma_{x}^{2}}\right)\right]}}$$
(14)

Note that Equation 14 for start-spread is very similar to Equation 11 for start-stop, with γ_{-} playing the role of γ_{u} . The correlations behave similarly, with

$$\begin{array}{c}
\rho(s_1, s_2) \\
\rho(s_1, d)
\end{array} \longrightarrow \begin{cases}
1 \text{ as } \sigma_b \to 0 \\
-1 \text{ as } \gamma_x \to 0.
\end{cases} (15)$$

These limits are approached at very different rates, however. Notice that the definition of γ_{-} in Equation 13 means that generally, $\gamma_{-} \gg \gamma_{u}$, which permits $\rho(s_{1},s_{2}) > 0 > \rho(s_{1},d)$. For example, the means of the data in Figure 7 are $r(s_{1},s_{2})=.34$ and $r(s_{1},d)=-.48$. These are readily accommodated by $B\sim .2$, $\sigma_{b}\sim .15$ and $\gamma_{x}\sim .2$ (γ_{l},γ_{u} in the neighborhood of .19, .13). These values are common in past applications.

It should be noted that the data may strain this model quantitatively if these two correlations are both large in absolute value. The data (Figure 7) do not show extreme values and hence are accommodated, although more extensive data sets may force modifications in SET (and the quasi-serial model, see below).

Serial Models

The serial and quasi-serial models in Table 3 are analyzed in an analogous fashion. The serial model calculations are immediate. The quasi-serial model has $s_1 = t + i_1$, $s_2 = t + h + i_2$, and $d = h + i_2 - i_1$. Hence, if $i_1 = i_2$, d = h and so $\rho(s_1,d) = 0$, disqualifying this variant as in the strictly serial model. Note also that $\rho(s_1,d) \to 0$ if both $\sigma_i \to 0$, because this too reverts to the strictly serial case.

Thus i_1 and i_2 must be independent samples. The correlations are then obtained in exactly the same manner as above, using Equations 6 and 8 for the variance and covariance of sums of random variables. The calculations are straightforward and need not be detailed here.

Notice that the equal instructional variances, $\rho(s_1,d) \to -1/\sqrt{2}$ and $\rho(s_1,s_2) \to 0$ as $\sigma_t \to 0$. The data may strain this quantitative feature somewhat, because subjects with large negative s_1,d correlations may not necessarily have small s_1,s_2 correlations.